

PULMONARY TUBERCULOSIS SEQUELAE: SPECTRUM OF RADIOLOGIC FINDINGS AND REVIEW OF LITERATURE

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Abstract:

Pulmonary tuberculosis (TB) is a common worldwide infection and a medical and social problem causing high mortality and morbidity, especially in developing countries. A variety of sequelae and complications can occur in the pulmonary and extra-pulmonary portions of the thorax in treated or untreated patients. In this article, we aimed to review the characteristic imaging findings of various sequelae of thoracic tuberculosis affecting the lung parenchyma, airways, vessels, mediastinum, pleura, and chest wall according to the experience of the department of radiology at the Hassan II^d University Hospital in Fes, Morocco.

Keywords: Pulmonary tuberculosis, radiologic findings, sequelae.

INTRODUCTION

Pulmonary tuberculosis (TB) is a common worldwide infection and a medical and social problem causing high mortality and morbidity, especially in developing countries. In accordance with the virulence of the organism and the defenses of the host, tuberculosis can occur in the lungs and in extra-pulmonary organs. A variety of sequelae and complications can occur in the pulmonary and extra-pulmonary portions of the thorax in treated or untreated patients.

In this article, we aimed to review the characteristic imaging findings of various sequelae of thoracic tuberculosis affecting the lung parenchyma, airways, vessels, mediastinum, pleura, and chest wall.

MATERIAL AND METHODS

We report a retrospective study that evaluates 105 verified cases of pulmonary tuberculosis presented at the Hassan II^d University Hospital with a variety of thoracic sequelae seen during the five year-period from January 2010 to December 2014. We included all patients who were treated for tuberculosis and presented with symptoms of cough or fever, hemoptysis, weight loss or loss of appetite and other chest complains such as chest pain and breathlessness. Patients who were found to be HIV-seropositive were excluded from the study. All our patients underwent chest radiograph and CT scan. Radiological lesions were classified into parenchymal lesions, airway lesions, vascular, mediastinal, pleural and chest wall lesions depending on the dominant chest CT scan picture visualized. A literature review was made using the Medline search.

RESULTS

The mean age of included patients was 55 years old. Male gender was prominent and the sex-ratio was 2.5: 1.

Chest CT scan showed various thoracic sequelae of tuberculosis :(Table I)

- In lung parenchymal lesions, there were cicatrization atelectasis in 20 cases, residual thin-walled cavities in 15 cases, destruction of lung in 6 cases, aspergilloma in 5 cases and bronchogenic carcinoma in 2 cases.
- Bronchiectasis (n= 20) was most commonly found in airway lesions followed by tracheobronchial stenosis (n=3) and broncholithiasis (n= 2).
- Mediastinal lesions were observed in 11 of the total cases, these consist of lymph node calcification in 8 cases, pericardial tuberculosis in 2 cases, fibrosing mediastinitis in 1 case.
- 11 patients had residual pleural lesions with 5 cases of pleural thickening, 4 cases of fibrothorax and 2 cases of pneumothorax.
- In Chest wall lesions; there were tuberculous spondylitis in 7 cases and empyema necessitatis in 1 case.
- Only 2 patients had vascular lesions of secondary pulmonary hypertension

Table I: type, number and percentage of thoracic sequelae of Tuberculosis in our study

Type of thoracic sequelae	Number of cases	Percentage
Parenchymal lesions		
• cicatrization atelectasis	20	19.04 %
• residual thin-walled cavities	15	14.28 %
• destruction of lung	6	5.71 %
• aspergilloma	5	4.76 %
• bronchogenic carcinoma	2	1.90 %
Airway lesions		
• bronchiectasis	20	19.04 %
• tracheobronchial stenosis	3	2.85 %
• broncholithiasis	2	1.90 %
Mediastinal lesions		
• lymph node calcification	8	7.61 %
• Pericardial Tuberculosis	2	1.90 %
• fibrosingmediastinitis	1	0.95 %
Pleural lesions		
• chronic empyema	5	4.76%
• fibrothorax	4	3.80 %
• pneumothorax	2	1.90 %
Chest wall lesions		
• tuberculous spondylitis	7	6.66 %
• empyema Necessitatis	1	0.95 %
Vascular lesions		
secondary pulmonary hypertension	2	1.90 %

DISCUSSION

Parenchymal lesions:

Residual Thin-walled Cavity

Residual thin-walled cavities may be seen in both active and inactive disease. After antituberculous chemotherapy, the tuberculous cavity may disappear; occasionally, the wall becomes paper-thin and an air-filled cystic space remains [1]. (**Fig. 1**)

The wall of a chronic cavity varies from 1 cm to less than 1 mm in thickness and may be smooth, sometimes simulating an emphysematous bulla. It can be difficult to distinguish true cavities from bullae, cysts, or pneumatoceles [2, 3]. (**Fig. 2**)

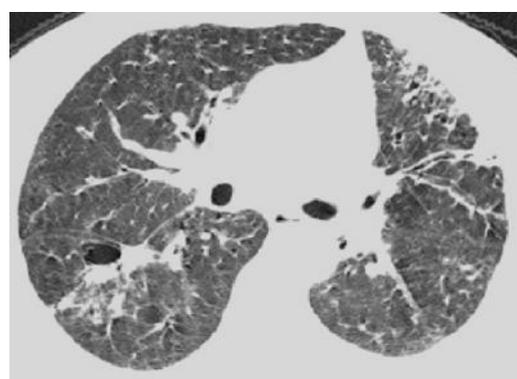


Fig. 1: Axial CT scan shows a thin-walled cavity, in the both upper lobes, stable for 2 years

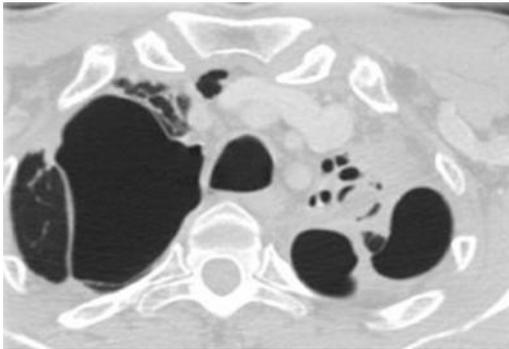


Fig. 2: High-resolution CT scan shows a thin-walled cavity, in the right lower lobe, which was stable for 3 years



Fig. 4: High-resolution CT scan shows parenchymal bands (blue arrow) with traction bronchiectasis (red arrow).

Cicatrization and Destruction of lung

Cicatrization atelectasis is a common finding after post primary tuberculosis. Up to 40% of patients with post primary tuberculosis have a marked fibrotic response, which manifests as atelectasis of the upper lobe, retraction of the hilum, compensatory lower lobe hyperinflation, and mediastinal shift toward the fibrotic lung [1, 4]. (Fig. 3)

A non-specific radiologic pattern of fibrosis consisting of parenchymal bands, fibrotic nodules and cavities, or traction bronchiectasis is occasionally encountered [5]. (Fig. 4)

Complete destruction of a whole lung or a major part of a lung is not uncommon in the end stages of tuberculosis. Such damage results from a combination of parenchyma and airway involvement. It may result from a progressive primary infection or from post-primary tuberculosis with a prolonged process of cavitation, spread to new areas, and subsequent fibrosis [6]. (Fig. 5).



Fig. 3: High-resolution CT scan shows atelectasis of the right upper lobe (blue arrow), with retraction of the hilum and traction bronchiectasis (red arrow).

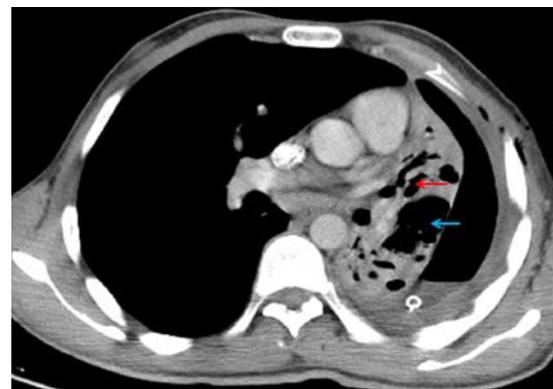


Fig. 5: Contrast-enhanced CT scan shows marked shifting of the mediastinum toward the left side. The left lung is destroyed and contains cavities (blue arrow), with traction Bronchiectasis (red arrow).

Aspergilloma

The prevalence of aspergilloma associated with chronic tuberculosis has been reported to be 11% [1]. The natural history of aspergilloma is variable. Hemoptysis is the commonest mode of presentation, with an incidence of around 80%, which is life threatening in 30% [7]. Aspergilloma is usually located within a cavity or ectatic bronchus and consists of masses of fungal hyphae admixed with mucus and cellular debris [8,9]. At radiography, a mobile, rounded mass surrounded by a crescentic air shadow is noted inside a lung cavity (Air-crescent sign or Monod sign) (Fig. 6A).CT demonstrates a mobile fungus ball, usually with air interspersed between the masses of mycelia (Fig. 6B, 7). Calcification of the mycelial ball occurs in some case [8, 9]. Thickening of the walls of tuberculous cavities or of the adjacent pleura is reported to be an early radiographic sign [1].



Fig. 6: Aspergilloma within a cavity in a 45-year-old man. (A) Frontal scout view of the chest shows a mass of soft-tissue opacity (red arrow) with an air-crescent sign (blue arrow) in the left upper lobe. (B) Contrast-enhanced CT scan shows a low-attenuation soft-tissue mass (red arrow) within the cavity, along with the air-crescent sign (blue arrow).

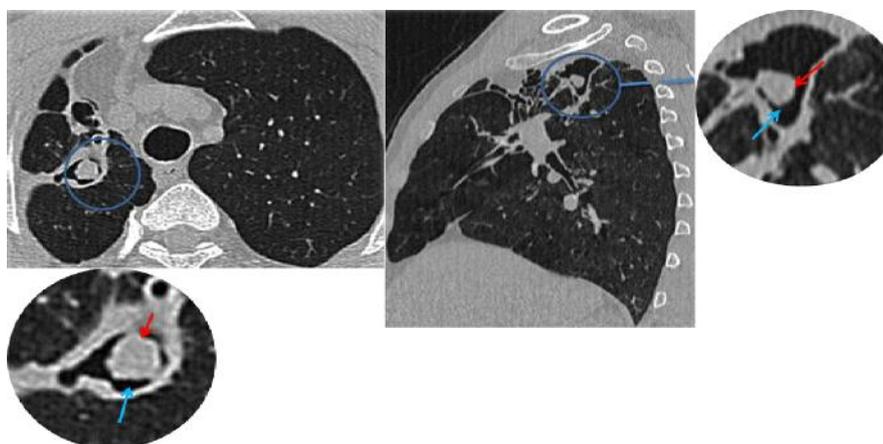


Fig. 7: Aspergilloma within a cavity in a 53-year-old man. Axial and coronal CT scan show cavity with a central soft tissue attenuating rounded mass (red arrow) surrounded by a Monod sign (blue arrow).

Bronchogenic carcinoma

Bronchogenic carcinoma and pulmonary tuberculosis often coexist, creating a difficult diagnostic problem. Manifestations of carcinoma may be obscured or misinterpreted as progression of tuberculosis.

Tuberculosis may favor the development of bronchogenic carcinoma by local mechanisms (scar cancer), or tuberculosis and carcinoma may be coincidentally associated. In addition, carcinoma may lead to reactivation of tuberculosis, both by eroding into an encapsulated focus and by decreasing the patient's resistance [10, 11]. (Fig. 8)

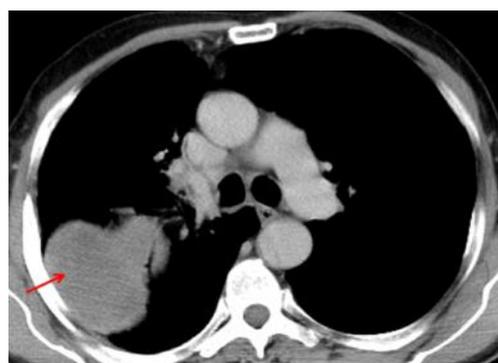


Fig. 8: Bronchogenic carcinoma with postprimary tuberculosis. Contrast-enhanced CT scan shows a lobulated mass in the apical segment of the right lower lobe (red arrow).

Airway Lesions

Bronchiectasis

Bronchiectasis may develop as a result of tuberculous involvement of the bronchial wall and subsequent fibrosis. Bronchiectasis is seen in 30%-

60% of patients with active post-primary tuberculosis and in 71%-86% of patients with inactive disease at high-resolution CT [12, 13]. Commonly, it occurs by destruction and fibrosis of the lung parenchyma with secondary bronchial dilatation (traction bronchiectasis) [1, 12, 14] (**Fig. 3, 5, 9**), but it may also be due to central bronchostenosis and distal bronchial dilatation [15]. Bronchiectasis located in the apical and posterior segments of the upper lobe is highly suggestive of a tuberculosis origin. When multiple cavities are encountered, the possibility that cystic bronchiectasis is present in addition to necrotic cavities, must be considered [1, 12, 14]. (**Fig. 10**)

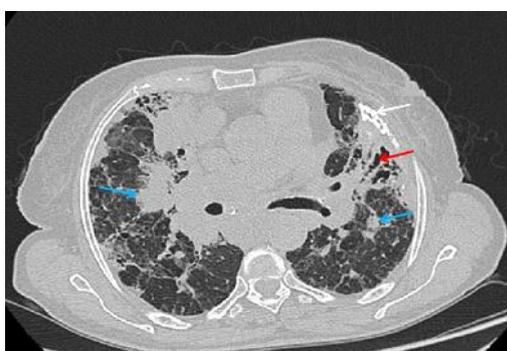


Fig. 9: Traction bronchiectasis in a 69-year-old man. Axial CT scan shows dilatation of bronchi (red arrow) within the right upper lobe with diffuse parenchymal fibrosis (blue arrows) and calcified pleural plaques (white arrow).



Fig. 10: Traction bronchiectasis in a 49-year-old man. Axial CT scan shows cystic bronchiectasis (red arrows) within the right upper lobe.

Tracheobronchial Stenosis

Tracheobronchial stenosis is not a frequent complication of pulmonary TB. Factors in pathogenesis include implantation of mycobacteria in the airway from a parenchymal lesion, direct

infiltration from an adjacent node, and peribronchial extension through lymphatic drainage or haematogenous spread. Stenosis may arise from extrinsic compression, from enlarged peri-bronchial lymph nodes or from excessive inflammatory and fibrous reaction affecting the airway wall [16]. The CT findings include concentric narrowing of the lumen, uniform thickening of the wall, and involvement of a

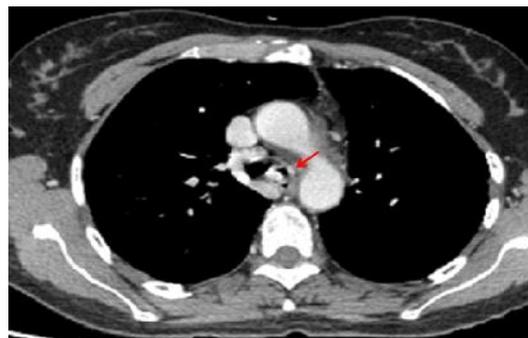


Fig. 11: Tracheobronchial stenosis in a 45-year-old man. Contrast-enhanced CT scan shows concentric narrowing of the left main bronchus (red arrow) with uniform thickening of the wall.

Broncholithiasis

Broncholithiasis is an uncommon complication of pulmonary tuberculosis and is defined as the presence of calcified or ossified material within the lumen of the trachea-bronchial tree. Presented symptoms may include cough, hemoptysis, wheezing, or evidence of recurrent pneumonia [18]. (Fig. 12, 13)



Fig. 12: Broncholithiasis in a 48-year-old man. Axial CT scan (mediastinal windowing) shows a broncholith (red arrows) within bronchus in the right lower lobe.

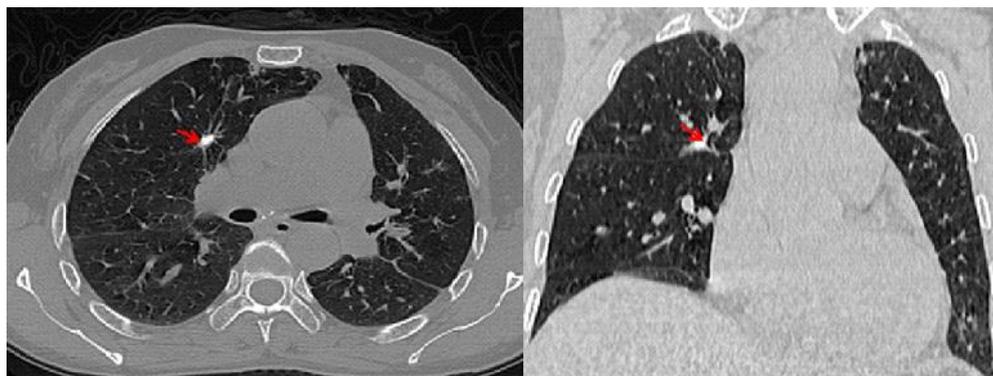


Fig. 13: Broncholithiasis in a 45-year-old man. Axial and coronal CT scan (lung windowing) show a broncholith in segmental bronchus (red arrow) within the right upper lobe

Vascular lesions:

Hypertrophied bronchial arteries

Bronchial arteries may be enlarged in bronchiectasis associated with tuberculosis or in parenchymal tuberculosis itself [19].

In patients with bronchiectasis, nodular and tubular structures with an appearance unlike that of lymph nodes or normal vessels in the mediastinum and around the central airway on high-resolution CT scans are suggestive of hypertrophied bronchial arteries. Recognition of this finding is important so that the bronchoscopist will not biopsy the hypertrophied bronchial arteries protruding into the airway lumen [19].

Rasmussen aneurysm presenting with haemoptysis

Rasmussen aneurysm is a rare phenomenon caused by weakening of the pulmonary artery wall from adjacent cavitary tuberculosis. Hemoptysis is the usual presenting symptom and may be life-threatening when it is massive [20].

There is progressive weakening of the arterial wall which occurs as granulation tissue replaces both the adventitia and the media. This is, then, gradually replaced by fibrin, resulting in thinning of the arterial wall, pseudo aneurysm formation, and subsequent rupture with haemorrhage [20].

Secondary pulmonary hypertension (PH) (Fig. 14)

The mechanism of development of PHT in treated TB patients is thought to result from residual pulmonary structural damage and pulmonary function abnormalities leading to gas exchange abnormalities and chronic hypoxia [9, 18]. It has also been suggested that repeated secondary respiratory tract infections, caused by residual chest x-ray abnormalities, play an important role in the

pathogenesis of PH in treated TB patients [21]. Though there is not a unique subgroup within the PH classification system, TB-associated PH is similar to group 3 of PH disorders [22].



Fig. 14: Secondary pulmonary hypertension in a 44-year-old man who was treated for pulmonary tuberculosis. Axial CT scan shows dilatation of the pulmonary artery with interlobular septal thickening (red arrows) and pleural effusion (blue arrow).

Mediastinal lesions:

Lymph Node Calcification

Tuberculous mediastinal lymphadenitis is a frequent manifestation of primary pulmonary tuberculosis. It is caused by the formation of tuberculous caseating granulomas in lymph nodes, which more commonly involves the right side. In the active stage, the nodes have central low attenuation and peripheral rim enhancement at CT, which correspond to caseation or liquefaction necrosis and granulation tissue with inflammatory hypervascularity, respectively, at pathologic analysis. With treatment, the nodes change in appearance, first becoming homogeneous and finally disappearing or resulting in a residual mass composed of fibrotic tissue and calcifications without low-attenuation areas [23, 24]. (Fig. 15)

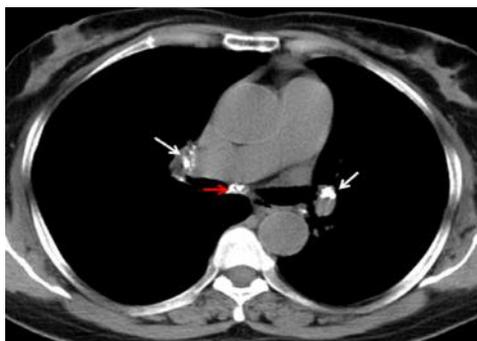


Fig. 15: Lymph node calcification in a 39-year-old man who was treated for primary pulmonary tuberculosis. Unenhanced CT scan shows a hilar (white arrows) and subcarinal (red arrow) lymph nodes with calcification.

Pericardial Tuberculosis

Tuberculous pericarditis is reported to complicate up to 1% of cases of tuberculosis [25]. Pericardial involvement is commonly caused by extra-nodal extension of tuberculous lymphadenitis into the pericardium. The pericardium can also be involved in miliary spread of the disease. At CT, lymphadenopathy and pericardial thickening with or without effusion may be seen [6]. (Fig 16)

Constrictive pericarditis occurs in about 10% of patients with tuberculous pericarditis. It is characterized by fibrous or calcific constrictive thickening of the pericardium, which prevents normal diastolic filling of the heart [26].



Fig. 16: Tuberculous pericarditis in a 45-year-old man. (A) Contrast-enhanced CT scan shows slight thickening of the pericardium (white arrows) with a marked amount of fluid in the pericardial cavity (arrowhead). (B) Contrast-enhanced CT scan shows enlarged lymph nodes (arrows) in the right lower paratracheal (red arrow) and Para-aortic (blue arrow)

Fibrosing Mediastinitis

Tuberculous mediastinitis (defined as infection and abscedation of mediastinal fatty tissue is extremely rare. In the actual era of antituberculous chemotherapy there are only few cases reported in the recent English-language literature [27]; it progresses insidiously and may result in mild symptoms, including cough and low-grade fever, and symptoms due to compression of the superior vena cava, esophagus, and tracheobronchial tree [28].

The CT findings include a mediastinal or hilar mass, calcification in the mass, trachea-bronchial narrowing, pulmonary vessel encasement, superior vena cava obstruction, and pulmonary infiltrates [28].(Fig. 17)

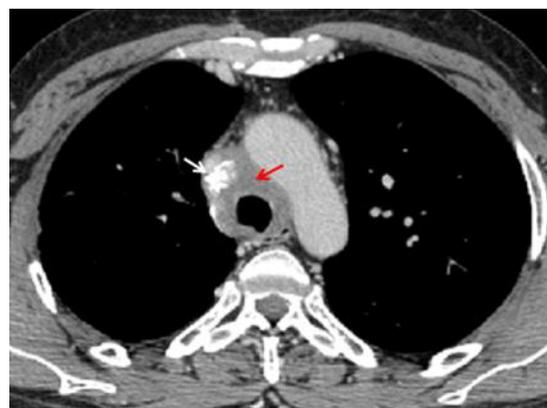


Fig. 17: Fibrosing mediastinitis in a 47-year-old woman. Axial contrast-enhanced CT scan shows a soft-tissue infiltration adjacent to the trachea and the aorta (red arrow), containing calcification (white arrow).

Pleural lesions:

Chronic Empyema

Pleural infection is usually caused by rupture of a sub-pleural caseous focus into the pleural space; less commonly, it is caused by hematogenous dissemination and contamination by adjacent infected lymph nodes. Tuberculous pleurisy progresses to become chronic tuberculous empyema, which may be defined as persistent, grossly purulent pleural fluid containing tubercular bacilli. In chronic tuberculous empyema, CT scans show a focal fluid collection with pleural thickening and calcification and with or without extra-pleural fat proliferation [29]. (Fig. 18)



Fig. 18: Chronic empyema, fibrothorax and pneumothorax in a 42-year-old man. Axial contrast-enhanced CT scan shows a fluid collection (red arrows) in the left hemithorax with visceral and parietal pleural thickening and calcification (blue arrow) relevant to chronic empyema and fibrothorax. There is pleural thickening with calcification and pneumothorax in the left hemithorax (white arrow).

Fibrothorax

Pleural thickening and fibrothorax tuberculous pleuritis often leave sequelae ranging from minimal pleural thickening, seen as obliteration of the costophrenic sulcus, to severe thickening, seen as fibrous tissue and calcification encompassing and restricting the lung and referred to as fibrothorax. Fibrothorax may be associated with extensive volume loss of the ipsilateral lung and even with ventilatory impairment. On radiography and CT, evidence of underlying parenchymal disease, extensive calcification of the fibrothorax, and unilateral involvement are strongly suggestive of previous tuberculosis [30]. Fig. (18, 19)

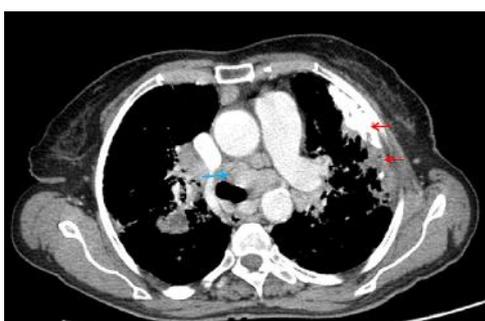


Fig. 19: Fibrothorax in a 50-year-old man. Axial contrast-enhanced CT scan shows visceral and parietal pleural thickening with calcification in the left hemithorax (red arrows). There are mediastinal lymph nodes (blue arrow).

Pneumothorax

Pneumothorax, secondary to tuberculosis, often heralds severe and extensive pulmonary involvement by the infectious process and the onset of bronchopleural fistula and empyema. It occurs in approximately 5% of patients with post-primary

tuberculosis. The pathogenesis involves pleural caseous infiltrates that undergo liquefaction resulting in pleural necrosis and rupture. If any apical abnormality is seen after reexpansion of a spontaneous pneumothorax, active tuberculosis should be considered [31, 32]. (Fig. 18, 20)

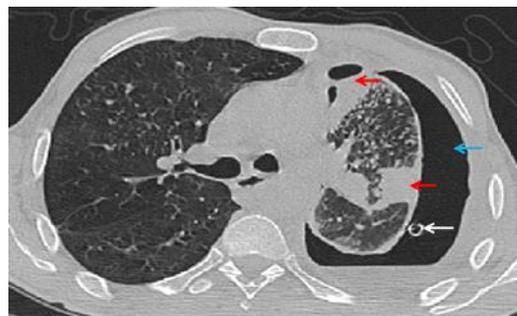


Fig. 20: Hydropneumothorax in a 50-year-old man after antituberculous chemotherapy. Axial CT scan shows areas of consolidation in left upper lobe (red arrow). A hydropneumothorax is noted in the left hemithorax (blue arrow) and was treated with insertion of a thoracostomy tube [white arrow].

Chest Wall Lesions:

Empyema Necessitatis

Empyema necessitatis, another well-known, is formed by breakage of the tuberculous empyema through the parietal pleura for spontaneous discharge of its contents. The most common site of empyema necessitatis is subcutaneous tissue of the chest wall, but other sites include the esophagus, breast, retroperitoneum, flank, groin, pericardium, and vertebral column. CT findings include well-demarcated, thick-walled fluid collections in intra-thoracic and extra-thoracic locations. A fistulous track between a pleural fluid collection and an extra-thoracic fluid collection is often revealed on CT [33]. (Fig. 21)



Fig. 21: Chest wall abscess in a 47-year-old man. Contrast-enhanced CT scan shows a mass with low attenuation and peripheral enhancement in the anterior chest wall (red arrow). The abscess has an intracostal portion (blue arrow). Excision of the mass was performed

Tuberculous Spondylitis

Tuberculous spondylitis is caused primarily by hematogenous spread of pulmonary infection and most commonly affects the lower thoracic and upper lumbar spine. The early radiographic manifestations of spinal involvement consist of irregularity of the vertebral end plates, decreased height of the intervertebral disk space, and sclerosis of the adjacent bone. With progression of disease, there is a tendency toward anterior wedging of the vertebral body, leading to kyphosis and development of a paravertebral abscess. CT scan demonstrates paravertebral abscesses with peripheral rim enhancement and low-attenuation centers after enhancement [34, 35]. (Fig.22)

MRI is preferred to plain radiography, CT, and nuclear medicine studies in detecting abnormalities of the bone marrow and soft tissues because it is more sensitive[36, 37].

Changes in both T1- and T2-weighted images are mainly due to the increased water content early detection of the pathological process [38].

The MR appearances of spinal tuberculosis are of high signal intensity on T2 and STIR sequences and low signal on T1 weighted sequences. On T1 weighted gadolinium enhanced sequences the abscess walls enhance uniformly and brightly; central necrosis is depicted as low signal intensity [39- 41]. (Fig. 23)

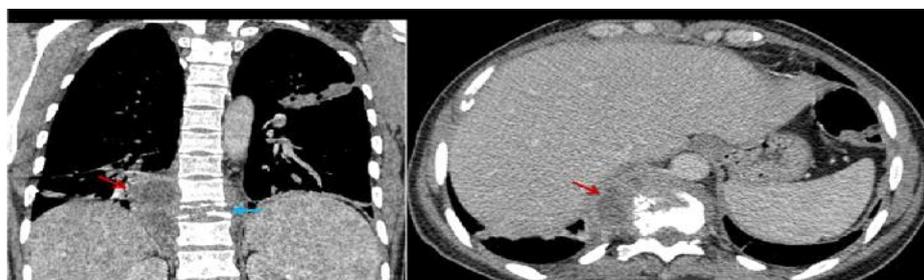


Fig. 22: Tuberculous Spondylitis in a 37-year-old man. Contrast-enhanced CT scan demonstrates collapse of T10 vertebral body (blue arrow) with paraspinal abscess (red arrow).



Fig. 23: Tuberculous spondylodiscitis in a 47-year-old man. MRI shows a partial destruction of T3-T4 vertebral bodies (blue arrows) with heterogeneous enhancement after gadolinium administration. Abscess is present in T3-4 disk space (red arrow) extending to paraspinal (yellow arrow) and anterior epidural spaces (green arrow).

CONCLUSION

In spite of all advances in anti-tuberculosis therapy, pulmonary tuberculosis can still originate important thoracic sequelae involving the lungs, airways, vessels, mediastinum, pleura, and chest wall. It is imperative that radiologists and clinicians understand the spectrum of these sequelae and complications to facilitate diagnosis.

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